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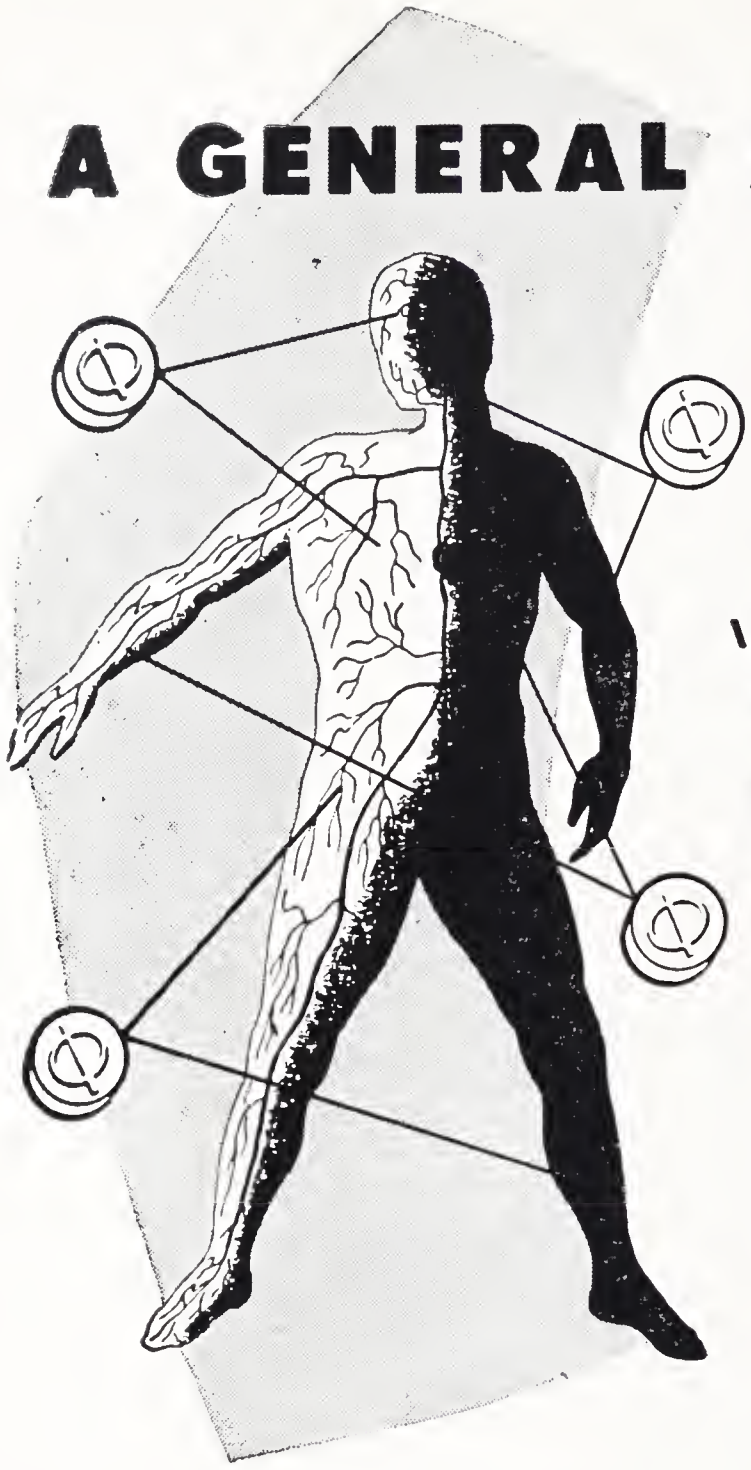

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EDITORIAL

Emergencies in the Practice of Medicine

In this issue several articles dealing with some of the aspects of Emergencies in the Practice of Medicine are presented. This subject encompasses not only the dramatic situation wherein a life is suddenly jeopardized, but also any situation where only immediate diagnosis and treatment can restore the health of the patient. It is impossible to cover such a large and complex field in so little space, but perhaps by emphasizing this facet of medicine the student may be encouraged to examine it more thoroughly.

In dealing with the patient, the ability to differentiate between the real emergency and the usual case is of prime importance, for on this choice lies the further conduct of the case and the future of the patient's health. It has been said that it is not the sins of commission, but the sins of omission, which cause most of the trouble in the practice of medicine. Therefore, the student must remember that he will have to deal with emergencies and he must be watching for them.

Once the doctor recognizes an emergency, he must rely on a previously well organized and practical knowledge of medicine and he must have the calmness to apply it quickly and effectively. This is very necessary, for the doctor in this situation seldom has time for consultation or for textbooks, and he may be hampered by poor location and by a lack of equipment. Even the interne, in a well equipped general hospital, is at a disadvantage, owing to a lack of experience and often to a lack of a source of immediately available advice. Thus, in this type of situation, a doctor is completely dependant on his own immediate ability and knowledge. The student can do a great deal to prepare himself for such a situation by thinking about the subject beforehand and by laying out a plan of action for dealing with the common emergencies he may meet. This will also help him to remain calm and effective at that time.

It should be pointed out that, although true emergencies are fairly rare, they are much more dangerous to treat. In the normal practice of medicine, the doctor usually has time to revise his diagnosis and/or treatment if the patient's condition warrants it. This is not true in the management of emergencies, so, although the doctor should be quick, he must also be very sure.

The best treatment is prophylaxis and the student should make a point of remembering well the many procedures in medicine which are potentially very dangerous and he must be prepared for any complications that may arise. Although the complications materialize only rarely, it is even more important that he remember them for otherwise he may grow careless with the original procedure and thus cause a higher incidence of complications and embarrassing emergencies than is necessary.

We hope that this issue will give you some insight into these problems and will encourage you to further examine this subject.

M. F. M.

THE MANAGEMENT OF OBSTETRICAL EMERGENCIES

R. J. FOOTE, 5T7

A. Emergencies with Bleeding

The conditions causing hemorrhage in obstetrical practiee may be divided into early – 0–28 weeks, and late – 28–40 weeks. The principles of management are the same in all cases:

1. Accurate diagnosis
2. Combat shock and blood loss.
3. Definitive treatment of the particular condition

I. early hemorrhage 0–28 weeks

(a) *Spontaneous abortion*

These cases present with a bloody discharge from the vagina and other symptoms, the degrees of which depend on how far the abortion has advanced.

Threatened abortion is the early stage. There is a bloody discharge, usually slight in amount without pain. There is no dilatation of the eervix nor expulsion of the products of conception. Inevitable abortion is the next stage. There are copious vaginal bleeding and crampy lower abdominal pain; the cervix is usually dilated. Inevitable abortion passes on into imminent abortion in which the pains become very strong and regular, like labour pains. With the expulsion of the products of conception the abortion beeomes eomplete, if all the products are expelled, or incomplete, if a part of the products of conception is retained. In incomplete abortion there is hemorrhage owing to retention of part of the placenta. Complete abortion however has the important difference that onee the products of coneeption are expelled the pains and bleeding progressively diminish.

Management:

(1) Diagnosis. In cases of abortion, threatened abortion must be differentiated from inevitable abortion or the other late stages. The diagnosis is based on the condition of the cervix, the presence of pains and their degree, and whether the products of conception have been passed.

(2) Combat shock and replace blood. The general surgical rules governing blood transfusion and shock therapy apply in all cases of abortion.

(3) Definitive treatment. In cases of threatened abortion eonservative therapy is recommended. The patient is put to bed and earefully observed. Morphine gr. 1/6 to 1/4 may be given. Progesterone 10 mg. and Vit. E 50 mg. are advocated, although there is no good evidence that they are effective. It is advisable not to give enemas or strong cathartics for several days.

In the other stages of abortion the treatment is the same for all cases. Transfusion if necessary is the first consideration. Hemorrhage is controlled with ergometrine 0.5 mg. or pituitrin 0.5 e.e. and an analgesic is given, morphine gr. 1/6 to 1/4. The patient is then prepared for the operating room where a curettage is performed if some of the products of conception are retained.

In the case of a septic abortion *all* manipulative procedures are kept to a minimum. Blood transfusion is advocated and antibiotic therapy started at once.

(b) *Ectopic pregnancy*

Tubal pregnancies with rupture present usually with a sudden severe unilateral pain, sometimes accompanied by vertigo and fainting. Vaginal bleeding may or may not occur. Shock occurs only in 10–20% of cases. Examination usually reveals abdominal tenderness and tenderness on moving the cervix or palpating the adnexa. Fifty per cent of cases have a boggy mass on one or other side of the uterus. A rapid pulse is a very constant sign. The blood pressure falls in proportion to the blood loss. The temperature in the absence of infection is rarely over 100–101.5°F.

If the pain and hemorrhage occur later (2–4 months) and the vaginal bleeding is very profuse, the possibility of interstitial pregnancy must be considered.

Management:

(1) Diagnosis. In the diagnosis of ectopic pregnancy with rupture, the following conditions must be considered:
   (a) Pelvic inflammatory disease
   (b) Abortion
   (c) Torsion of an ovarian cyst
   (d) Acute appendicitis
   (e) Rupture of a follicular cyst or corpus luteum with intra-abdominal hemorrhage.

(2) Blood transfusion is essential.

(3) Definitive treatment. Morphine gr. 1/6 to 1/4 is given and the patient prepared for laparotomy. During the preparation time it is wise to place the patient in the Trendelenburg position. In the case of ruptured tubal pregnancies, after starting the blood transfusion, an interval should be left to combat shock before operating.

(c) *Bleeding due to hydatidiform mole*

Bleeding due to a mole may be slight or profuse. Usually the patient has passed portions of the mole with the blood. Occasionally the mole penetrates the uterus giving the signs and symptoms of abdominal hemorrhage and shock. Sometimes infection is present. The uterus is frequently enlarged more than is normal for a pregnancy of the same duration. This condition is frequently incorrectly diagnosed as a threatened or an inevitable abortion. Once vesicles have been passed the diagnosis becomes obvious.

Management:

(1) Diagnosis. Profuse or spotty bleeding with an abnormally enlarged uterus is suggestive of a mole but the existence of multiple pregnancies must be remembered. Passage of mole vesicles is, of course, diagnostic.

(2) Transfusion will be necessary if the bleeding has been profuse or if the mole has penetrated into the peritoneal cavity. It is best to have transfusion apparatus ready whether the bleeding is profuse or not.

(3) Definitive treatment is directed at expulsion of the mole. Pituitrin 0.5 c.c. in divided doses or a 0.1% pitocin drip and morphine gr. 1/4 are given. The uterus may then be evacuated with ovum forceps if the case is uncomplicated or curettage may be performed. Curettage must be done with extreme care since the uterus is very soft. If hemorrhage is not very severe or if there is infection, curettage should be deferred. If the uterus is grossly dilated or if the mole has perforated the

uterus, laparotomy or hysterectomy must be performed.

(d) *Traumatic injuries to the uterus*

These may occur owing to curettage or to attempts to induce an abortion. The uterus is perforated and there may be loops of intestine present which were dragged through the wound by the instrument. There is massive hemorrhage and shock.

Management:

(1) Diagnosis. The diagnosis is made on a history of attempted abortion or curettage. The history of criminal abortion may be difficult to obtain and time may not allow an examination or detailed questioning.
(2) Transfusion is required immediately and large quantities of blood should be available for immediate use.
(3) Definitive treatment consists of immediate preparation for laparotomy to allow a surgical repair of the uterus to be done. If the injury is due to criminal abortion, then chemotherapy consisting of massive doses of penicillin in combination with other antibiotics or sulfas should be started immediately.

II. LATE HEMORRHAGE 28–40 WEEKS

(a) *Placenta previa*

Any painless bleeding in the last trimester of pregnancy should be considered to be due to placenta previa until proven otherwise. The bleeding may be persistent or intermittent, profuse or scanty. However, the first occurrence of bleeding will rarely exsanguinate the patient.

Management:

(1) Diagnosis. Bleeding in the last trimester without pain or other symptoms is highly suggestive of placenta previa. Confirmation is made on X-Ray examination or, on vaginal examination, when a spongy mass is felt in a dilated cervix or a boggy mass in an undilated cervix.
(2) Transfusion must be done before anything else and blood must be available for ante or postpartum hemorrhage.
(3) Definitive treatment. Do not vaginally examine patients suspected of placenta previa except in an operating room prepared for Cesarian section. Vaginal packing should not be done.

Expectant treatment can be carried out in cases where the bleeding is slight and subsides promptly and the fetus is not well into the stages of viability. However, if the fetus is well into the stage of viability and the placenta previa of major degree the fetus should be delivered. The patient is therefore prepared for vaginal delivery (or for Cesarean section) if the cervix is dilated, and the placenta previa of minor degree. In either case adequate blood must be available.

(b) *Abruptio placentae*

The hemorrhage is often concealed in this condition. The patient usually presents with very severe pain, a fall in systemic blood pressure and a peculiarly ligneous or rigid uterus. Occasionally the uterus is also grossly enlarged. The hemorrhage may be external, in which case the condition may be confused with placenta previa.

Management:

(1) Diagnosis. Abdominal pain in the last trimester and the development of a ligneous uterus are almost pathognomonic of abruptio placentae. If there is external hemorrhage abruptio placentae

can be differentiated from placenta previa because of the presence of pain and the fact that a placenta cannot be demonstrated on vaginal examination.

(2) Transfusion must be started at once.

(3) Definitive treatment. Morphine gr. 1/4 should be given. If the membranes have not ruptured, low rupture should be performed when shock has been overcome.

(c) *Post-partum hemorrhage*

Post-partum hemorrhage may be primary, occurring within the first 24 hours, or it may be secondary, occurring after 24 hours. Hemorrhage of more than 500 c.c. is considered pathological. Bleeding occurs from lacerations of the uterus and/or birth canal or from the placental site due to atonia of the uterus. Late hemorrhage may be due to the retention of part of the placenta or bleeding from an area of necrotic decidua.

Management:

(1) Diagnosis. Generally speaking, if the uterus is tonic, the bleeding is due to a laceration. If the uterus is atonic, bleeding may be from the placental site, or from a laceration. The diagnosis of late hemorrhage due to placental retention is made by a vaginal examination and curettage.

(2) Transfusion is given in proportion to the blood loss.

(3) Definitive treatment. With early hemorrhage the first necessity is to stop the bleeding. This may be done by massaging the uterus. At the same time ergotamine 0.5 mg. or pituitrin 0.5 c.c. are administered I.M. Bimanual compression of the uterus or a tamponade may also be used to stop bleeding. Direct compression of a laceration is best done before suturing is attempted.

Hemorrhage due to retained placenta is treated by ergonovine 0.5 mg. followed by manual removal of the tissue of curettage.

(d) *Rupture of the uterus*

Traumatic or spontaneous ruptures of the uterus are rare occurrences. Traumatic ruptures are caused by manipulative obstetrical delivery or direct forceful violence as occurs in auto accidents. Spontaneous rupture is usually through a Cesarian scar. Either may be symptomless for some time. The symptoms are agonizing lower abdominal pain and then cessation of all uterine contractions, with relief of the pain. There may or may not be a bloody discharge. The blood pressure is decreased and there are other signs of shock.

Management:

(1) Diagnosis. This condition must be thought of in any case of severe pain and shock. Often there has been a change in the presenting part or the fetus cannot be felt on vaginal examination. The tear in the uterus may be felt sometimes.

(2) Transfusion. Transfusion must be started and continued throughout the operation.

(3) Definitive treatment. The definite treatment includes giving the patient both morphine gr. 1/4 and blood and preparing the patient for immediate laparotomy.

(e) *Inversion of the uterus*

After delivery, the uterus is turned inside out and the fundus comes to lie in the vagina. Shock is almost always present. The patient often cries out after the placenta is delivered and

bears down. This is usually followed by profuse hemorrhage.

Management:

(1) Diagnosis. The diagnosis is often made on direct examination. A mass is always felt in the vagina. Bearing down after the placenta is delivered followed by hemorrhage is usually very suggestive.
(2) Transfusion is required in large amounts.
(3) Definitive treatment. Morphine gr. 1/4 and Adrenalin 1:1000 0.5–1 c.c. are given at once. Then an attempt should be made to replace manually the placenta. After the uterus is replaced ergonovine 0.5 mg. or pituitrin 0.5 c.c. should be administered. If the uterus still tends to relax and fall back, the vagina can be packed with sterile gauze. If the manual replacement cannot be accomplished due to incarceration by the ring, surgical replacement may be necessary.

B. Emergencies without Bleeding

(a) *Preeclampsia or eclampsia*

The preeclamptic patient seen in the emergency department is usually in a very late stage of preeclampsia. She may complain of epigastric pain. The face and fingers are edematous and puffy. She may have visual disturbances. Blood pressure readings will show hypertension and ophthalmic examination will show retinal changes.

The frankly eclamptic patient is commonly seen in the fulminating stage. There may have been frank epileptiform convulsions. In occasional cases the patient is in a profound coma. Ninety per cent of cases show edema and all have proteinuria and hypertension.

Management:

In both cases the patient should be placed in a dim room in bed. Never leave these patients alone.
Control of irritability is the most important consideration of treatment. In the preeclamptic patient this may be done with Sodium Luminal gr. ii I.M. In the eclamptic patient the method is to give Sodium Amytal gr. iii to viiss I.V. One hour later Sodium Luminal gr. ii I.M. should be given and may be repeated as necessary.
After the convulsions have been stopped, and only then, are the indications for terminating the pregnancy considered. Termination is either by surgical induction of labour or Cesarean section.

(b) *Abnormal presentations*

The first step is a diagnosis of the presentation. Before any steps can be taken a careful evaluation must be made as to whether the presentation is breech, transverse, brow, face or compound. The presentation is determined by physical examination and/or X-Ray. Also to be considered in cases of obstructed labour is the possibility of multiple pregnancy or a congenital abnormality of the fetus.

The second consideration is whether the fetus is alive or dead. The fetus must always be considered alive unless there is very strong evidence that it is dead, i.e., a uterus that is not of corresponding size with the duration of pregnancy or lack of fetal heart beat. Occasionally a mascerated skull can be palpated through the cervix.

Management:

Management is directed toward delivery of the child. If the cervix is fully dilated, vaginal delivery in attempted. If the cervix is not dilated, Cesarean section may be required to

achieve delivery. Manual dilation of the cervix leads only to lacerations. Destructive operations are very rarely attempted and only when it is known definitely that the fetus is dead.

(c) *Prolapse of cord*

Prolapse of the cord rarely affects labour insofar as the mother is concerned. It is, however, one of the chief causes of fetal mortality. The cord is compressed between the presenting part and the pelvic wall. It is diagnosed by seeing or feeling the cord on vaginal examination. This condition must be borne in mind whenever there is a sudden cessation of fetal heartbeat in an otherwise normal labour. Prolapse of the cord occurs often in cases with abnormal presentations and in cases with malformation of the fetus and these must be determined before delivery is attempted. The viability of the fetus must be determined before any operative delivery is attempted.

Management:

(a) If the child is dead, expectant treatment is followed. The dead fetus will deliver spontaneously.

(b) If the fetus is alive and viable or likely to survive, the patient must be prepared for immediate delivery as this is the only means of saving the child. If the cervix is dilated, vaginal delivery is the method of choice, aided by forceps, extraction or version as indicated. If the cervix is not dilated, Cesarean section must be performed.

Acknowledgment

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THE EYE IN THE EMERGENCY DEPARTMENT

J. R. WILKES, 5T6

ALL too frequently blindness results from a disastrous mistake due to overly hasty examination or ineffectual treatment in a hospital emergency department. This article is directed towards preventing such unfortunate mishaps.

A proper examination of the eye requires a good source of light and a thorough routine observation of all the anatomical components visible to the examiner.

A. Mechanical Injuries

1. lacerations of the eyelid

A horizontal tear not involving the lid margin is sutured with dermal nylon 5–0.

A vertical tear which involves the lid margin must be repaired with care, and it is most important that the first suture be at the lid margin restoring its continuity accurately. If this is not done the lid margin will be nicked, resulting in ineffectual lid function.

2. LACERATIONS INVOLVING THE CANALICULUS

A laceration involving the inner margin of the lid must be examined carefully for involvement of the canalieulus. If this has occurred the eye is covered with a sterile pad and the patient referred to an ophthalmologist.

3. FOREIGN BODIES

These are best classified as superficial, imbedded or intraocular. The history of the accident is a most useful aid in determining the site of the body.

Superficial: The superficial foreign body is usually caused by something from the street being blown into the eye. The treatment is to retract the lid, and often all that is required is to remove the body with some absorbent cotton on the end of an applicator. Sodium sulfaeetamide 10% may be applied.

Imbedded: The type of accident whieh causes an embedded foreign body may be exemplified by the entering into the eye of partieles from a spinning emery wheel. Pontoeaine ½% is instilled in the eye. A sharp spud is required, and a loupe is used to improve vision. The foreign body is removed and sodium sulfacetamide 10% ointment applied. The eye is eovered with a patch. However, any eye whieh is diseharging secretion must be left open and treated with hot compresses and sodium sulfacetamide 10% ointment. A determined effort should be made to get out any remaining rust.

Some foreign bodies such as gun powder and stone are well tolerated in the parenehyma onee the initial stages of irritation are past, and the eye does best if left alone. For initial treatment, irrigate the eye thoroughly with saline but do not dig for small pieees.

Intraocular: Any history of a partiele having entered the eye when the patient was using a hammer or some sueh instrument where a relatively heavy particle might enter the eye at high speed must be considered that of an intraocular foreign body.

The eye must be X-rayed to see whether a foreign body can be demonstrated. Any perforating injury of the eyeball, whether the foreign body is retained or not, requires the serviees of an ophthalmologist.

4. ABRASIONS OF THE CORNEA

Any non-penetrating mechanical trauma to the eye is liable to eause a corneal abrasion. The extent of the abrasion may be estimated by Fluoreseein stain, and Pontocaine ½% is added for pain. Bichloride of mercury salve 1:3000 is added and the lid immobilized with a well-fitting patch. The patient should return next day.

An abrasion caused by a baby's finger is treated as an ordinary abrasion for the first two or three days, but if it progresses to keratitis the patient must be treated with atropine 1% and referred to an ophthalmologist.

5. CONTUSIONS OF THE EYE

Ecchymosis of lid: The eyelids should be retraeted and the eyeball inspected. If serious injury is not present apply pressure bandages.

Subconjunctival hemorrhage: This clears up in seven to ten days and no treatment is required.

Traumatic mydriasis: This condition is charaeterized by irregularly shaped pupils due to rupture or paresis of the sphincter of the iris. There is no emergency treatment, but the patient must see an ophthalmologist.

Anterior chamber hemorrhage: The complieations of this lesion are secondary glaucoma and blood stain of the cornea and are caused by seeondary hemorrhage which is best avoided by early treatment. Complete bed rest and binocular patching are required. An ophthalmologist should be called.

Traumatic iritis: Circumcorneal injection, with a swollen, dull iris and small pupil associated with pain, photophobia, lacrimation and interference with vision are the hallmarks of this condition. Atropine 2% is instilled every two hours and an ophthalmologist is called.

Vitreous hemorrhage: This condition always indicates a serious eye injury; it takes at least three months to clear, leaving vitreous opacities from broken down vitreous structure. It is diagnosed by the absence of the red reflex. There is no emergency treatment and the patient is referred to an ophthalmologist.

Rupture of the sclera: This condition is diagnosed by the decreased intraocular tension, and, because most often the rupture is at the limbus, by the pupil being drawn towards the site of the rupture. The iris may also be prolapsed. At any rate, the injury is a most striking one, and the emergency treatment is to cover the eye and call an ophthalmologist immediately.

Rupture of the choroid and *detachment of the retina* are other serious injuries resulting from contusion.

Avulsion of the optic nerve: This condition is manifested by sudden and complete blindness. An ophthalmologist is called and there is no emergency treatment.

B. Chemical Injuries

These may be classified as:

1. NON-PROGRESSIVE. Acids, except hydrofluoric, sulfurous, and acids containing heavy metals.
2. PROGRESSIVE. Alkalies: Lime, Dye ($NaOH, KOH$), Ammonia (NH_4OH). Others: Certain acids; e.g. hydrofluoric, sulfurous and chromic.
3. DUE TO IRRITANTS.

EMERGENCY TREATMENT

In these lesions time is an all-important factor, as penetration is rapid and deep.

Lime is the commonest alkali causing chemical injury to the eye. It is most important to start copious irrigation of the eye immediately upon the patient's arrival. If boracic acid is present it may be added to neutralize the alkali. Topical anesthesia, Pontocaine ½%, is added after the irrigation, and the lids everted and a thorough search made for any material present, followed by further irrigation. Sodium sulfacetamide 10% ointment is applied. An ophthalmologist is called the moment the patient presents in the emergency.

Sulfur dioxide and ammonia, which are present in refrigeration plants and in other industries, are extremely harmful when in the eye, and usually the patient arrives at the emergency department too late to have his sight saved. The treatment is the same as that for lime.

Acid burns are common in garage mechanics, storage battery workers and photographers. Although these are more painful they are less damaging. They are treated much the same as the alkali burn, except that sodium bicarbonate is used instead of boracic acid. The ophthalmologist ought to be consulted, but not with the same urgency.

C. Injuries Due to Radiant Energy

1. ELECTRIC FLASH AND WELDING RAYS

These are highly concentrated ultraviolet rays which have an abiotic effect on the cornea.

Treatment is directed at relieving pain and at reassurance. Enable the patient to sleep and he will probably be asymptomatic upon awakening.

2. ECLIPSE BLINDNESS

This occurs when the eyes are improperly protected during an eclipse of the sun. It is due to the focusing of infrared rays on the macula. Any injury is permanent. The only treatment is pro-

phylactic: the public must be warned before an eclipse.

3. SNOW BLINDNESS

This is comparable to electric flash and arc welding burns, with the same treatment. Prognosis is invariably good.

4. X-RAYS AND RADIUM

Beta rays do not penetrate more than 1 mm. and are used therapeutically. Deeper penetrating gamma rays can cause cataracts. There is no treatment.

D. MISCELLANEOUS EMERGENCIES

It is conceivable that any ocular lesion may present in the emergency department, but space permits mention only of some of the commoner or more severe ones. In particular acute congestive glaucoma and acute iritis must be watched for, because if neglected blindness may result.

1. VASCULAR LESIONS

Retinal artery thrombosis or embolism: Treatment is of no avail unless the patient is seen within one hour of the accident. Within the hour vasodilators or paracentesis of the eyeball might be of some use. The greyish fundus and cherry-red fovea are characteristic. An ophthalmologist should be called the moment this picture is seen.

Retinal vein thrombosis: Sight is soon lost in this condition. Engorged thin veins with a hemorrhagic fundus are characteristic. There is no emergency treatment and the patient should be referred to an internist.

2. ACUTE CONGESTIVE GLAUCOMA

The onset of rapid failure of sight, and severe pain in the eye radiating along the fifth cranial nerve, sometimes causing nausea and vomiting, characterize this condition. The eye is red, with pronounced circumcorneal injection; the lids are swollen and edematous; the cornea is steamy, with shallow anterior chamber and dilated pupil.

Treatment must be carried out immediately, and consists of pilocarpine 2% or 4% and eserine sulphate ¼% alternate drops every ten minutes, and Diamox 250 mg. stat., and 250 mg. in two hours per os. Ice compresses may also be applied. The patient is reassured and kept at rest. The patient should be referred to an ophthalmologist.

3. ACUTE IRITIS

Pain, photophobia, lacrimation, and interference with vision are the most important symptoms of this condition.

The iris looks altered; it is swollen, dull, and its colour fades. There is circumcorneal injection and the pupil is contracted, sluggish and irregular.

Treatment must be immediate. It consists of atropine 2% every two hours for the first twelve hours, followed by every four hours until recovery is complete. Hot moist compresses are soothing, and bed rest in a dark room with aspirin or codeine to relieve pain are required. The patient should be referred to an ophthalmologist.

4. CORNEAL ULCERATION AND DEEP KERATITIS

The inflammatory lesions of the cornea are caused by invasion of micro-organisms, and as they heal by scar they must be checked with antibiotics.

In any history of a recent abrasion or foreign body a dull grey corneal infiltration must be watched for, in association with pain, photophobia and lacrimation.

5. SYMPATHETIC OPHTHALMITIS

If, within two weeks of a perforating injury to one eye, the other eye becomes inflamed, sympathetic ophthalmitis must be suspected. If this is missed the patient will go blind. A patient with sympathetic ophthalmitis must be referred to an ophthalmologist at once for enucleation of his perforated eye.

6. HERPES ZOSTER OPHTHALMICUS

If there is herpetic eruption along the ophthalmic branch of the fifth cranial nerve, the patient will develop iritis. Iritis is preceded by an eruption on the tip of the nose and requires the services of an ophthalmologist.

7. CONJUNCTIVITIS

An itching or smarting of the eye, associated with a red conjunctiva and mucoid secretion which holds the lids together in the morning characterizes this condition.

Warm compresses three times a day, irrigation with boric acid, and, most important, sodium sulphacetamide 10% three times a day, constitute the treatment.

8. DIPLOPIA

The onset of double vision is most often due to involvement of the third, fourth or sixth cranial nerves in some pathological process. It is essentially a problem for the internist and should be referred to him.

Treatment in the emergency department is to cover the deviated eye, which will relieve the patient's distress.

SUMMARY

The important ophthalmologic problems confronting the staff of a hospital emergency department have been presented and the emergency treatment outlined.

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RESUSCITATION OF THE NEWBORN

J. D. TRELFORD, 5T6

ONE of the greatest responsibilities a doctor has is the resuscitation of the newborn infant. The mortality in the immediate neonatal period has not significantly decreased as has the perinatal mortality over the past forty years despite the great advances in medicine. Thirty-five percent of deaths in the first year of life occur in the first twenty-four hours and fifty percent of these in the first hour. About fifty-nine percent of all these deaths are associated with anoxia. This review deals with some of the theoretical, scientific and practical aspects of this subject as presented in the literature over the past five years. The paper will be divided into physiological, pathological, and therapeutic aspects.

PHYSIOLOGICAL ASPECTS

I. FETAL BLOOD

Oxygenation of the fetus occurs through the placenta and varies directly with maternal circulation through the placenta and the oxygen in the mother's blood. The oxygenation of the fetus does not remain constant throughout pregnancy but falls gradually until the fortieth week at which time the baby is still adequately supplied.[1]

At birth it is interesting that certain blood chemistries are altered from those in the adult. The baby is acidotic at birth due mainly to excessive amounts of pyruvic and lactic acids, produced by anaerobic metabolism which allows the infant to survive anoxic periods. See table one.

Table One[2]

	Normal fetus at birth	Asphyxiated
Arterial 02 capacity	20.8 vol. %	20.8 vol. %
„ „ content	10.5 vol. %	1.8 vol. %
„ „ saturation	50.5 vol. %	6.5–4.6 %
CO2 tension	32 mm. Hg	65 mm. Hg
Lactic acid content	35 mg. %	85–90 mg.%
pH of the blood	7.35	7.05

II. EXPANSION OF THE LUNGS

1. Jaykka[3] recently has put forth a new theory based on his work using the lungs of stillborn babies in which no respiration could occur. He introduced the hypothesis that the expansion of the pulmonary alveoli is caused by capillary erection, which is effected by the initiation of pulmonary circulation. In the intrauterine stage the blood is shunted past the lungs so that only 5% of the total blood volume goes through the lungs. At birth with the closing of the foramen ovale, there is a sudden increase in the pulmonary circulation. Jaykka feels that it is this force that causes the lungs to expand. With this theory the ductus arteriosus and the foramen ovale are indispensable in preventing the initiation of blood flow through the pulmonary circuit and keeping the lungs collapsed before birth. He also postulates that this may be the expansile force causing the alveoli to open in cases of congenital laryngeal atresia.[4] He does not, however, explain the mechanism which closes the shunts on which his whole theory is based.

2. A baby on expanding its chest has been found to exert intrathoracic pressure changes of 60 cm. water with initial gasps which drops to about 24 cm. water in the breaths to follow. From this it will be noted that the initial inflation of alveoli requires more force than the subsequent respiration.

III. MECHANISM OF THE FIRST BREATH

Barcroft[5] from his observations of sheep embryo found that after the sixtieth day no respiratory movements occurred unless the fetus was anoxic. He goes on to describe four stages of intrauterine reaction to stimuli, which occurs after conception before the sixtieth day. From this he postulates that the baby on delivery starts to breathe, not as a continuation of intrauterine movements causing a flow of amniotic fluid through the lungs, but to the resulting asphyxia and stress at birth causing a reversal of development to an earlier stage of intrauterine respiration occurring before the sixtieth day.

IV. CONTINUATION OF RESPIRATION[6, 7, 8]

It has been concluded by most authors that the chemoreceptors of the carotid and aortic bodies play a most important part in respiration in the newly born. The exact stimulus and mechanism is, however, not entirely clear. It is believed that the stimulus for respiration may not be carbon dioxide per se, but may be due to the overall changes of pH of the blood.

Pathological Aspects

I. OXYGENATION OF THE BABY

Should the uterus be in tetany owing to dystocia or to other factors, the maternal blood will be unable to circulate through the placenta; should the uterus be flaccid, the fetal blood will

flow into the placenta severely depleting the infant's blood volume; in both cases the fetus will suffer from asphyxia.

It was noted that in the third trimester, when uterine contractions are present, a drop of maternal blood pressure below eighty for five minutes will cause fetal anoxia.[9]

In post maturity the oxygenation of the fetus falls rapidly until, in the forty-third week, the baby will have only enough oxygen to maintain a resting state. In order to compensate for the fall in oxygenation, the fetus will increase the oxygen carriage of its blood and may have a hemoglobin of 143% or more.

The ability of the fetus to survive anoxia, by the use of anaerobic metabolism, is severely reduced by a low maternal blood sugar and may be corrected by increasing the maternal blood sugar back to normal. This is important in dealing with diabetic mothers.

II. RESPIRATION

It has been noted in premature babies that, although there is sufficient lung tissue to adequately meet its metabolic needs, a high energy requirement is necessary to maintain respiratory function.[10]

III. LESIONS FOUND AT AUTOPSY

1. General

The fundamental lesion found is complete atelectasis, congestion, and edema of the lungs and hemorrhage into the lungs. Asphyxia also causes damage to all tissues and one finds interstitial edema, rupture of vessels and tissue necrosis particularly in the liver and brain.

2. Hyaline membrane disease

This is a pathological diagnosis recognized by the presence of an eosinophilic material lining alveoli and plugging small bronchioles, thus causing atelectasis. Cells are often seen in the middle of the alveoli. This condition is never found in the stillborn and rarely in postmature babies. It is commonly found in, infants of diabetic mothers, those born of Caesarean section and prematures. The origin and significance of this membrane is not known; many theories have been postulated.

Jaykka suggests that this membrane may be explained by the fact that the underdeveloped lungs are unable to stretch rapidly over the capillaries, which are erected by the initiation of the pulmonary circulation, so that the cuboidal epithelium lining the alveoli becomes detached and falls to the centre.

Hyaline membrane disease causes secondary atelectasis usually occurring in the second day but it may occur as early as one and one half hours after the initiation of respiration.

Steiner[11] divides this condition into three progressive clinical stages as diagnosed by X-ray.

First: Fine miliary mottling throughout the lung fields (probably diffuse alveolar collapse and consolidation).

Second: Appearance of coalescent areas of atelectasis proceeding to lobular and lobar consolidation with collapse.

Third: Lungs show total non-aeration. The bronchial tree may be seen.

In the series studied no child survived the third stage.

ETIOLOGY AND DIFFERENTIAL DIAGNOSIS OF ASPHYXIA

I. ETIOLOGY

1. Maternal factors

(a) Over 35 years of age.

(b) Primipara more than multipara to the eighth child.

(c) General health, especially: cardio-vascular disease, pulmonary disease, metabolic disease.

(d) Special conditions: diabetes, thyrotoxicosis, and eclampsia.

2. Fetal factors

(a) Congenital abnormalities, especially pulmonary and cardiovascular.

(b) Fetal disease; erythroblastosis foetalis, congenital syphilis, etc.

(c) Prematurity and postmaturity.

(d) Anoxia of placental origin, infarct, placenta previa with compression by the head, premature separation.

(e) Cord torsion, knotting or prolapse of cord, compression or rupture of velamentous insertion of cord.

3. Complications of labor.

(a) Types of delivery in order of severity: version, high forceps, breech extraction, section, and low forceps with episiotomy having the lowest incidence.

(b) Induction with pitocin or quinine increases the incidence of resuscitation.

(c) Disproportion.

(d) Uterine tetany or Bandel's contraction ring.

(e) Uterine rupture and hemorrhage.

(f) Drugs depressing respiration used as maternal analgesics.

II. DIFFERENTIAL DIAGNOSIS OF ASPHYXSIA

1. An airway full of mucus.
2. Atelectasis with or without hyaline membrane disease.
3. Persisting effect of narcotic drugs given to the mother.
4. Pulmonary immaturity.
5. Pneumonia.
6. Intrapulmonary hemorrhage.
7. Spontaneous pneumothorax and interstitial mediastinal emphysema.
8. Cardiovascular and other congenital abnormalities.
9. Oesophageal tracheal fistula.
10. Diaphragmatic hernia.
11. Intraventricular hemorrhage.
12. Cerebral irritation and raised intracranial pressure.

EVALUATION OF THE NEWBORN

Two systems are outlined here.

I. FLAGG'S CLASSIFICATION[12]

1. Stage one: "Depression"—respiration, slow and irregular—tendency to cyanosis—reflexes depressed—capable of being aroused.
2. Stage two: "Spasticity"—respiration irregular, gasping and shallow—cyanotic—reflexes sluggish—glottic reflex present on suctioning.
3. Stage three: "Flaccidity"—respiration none—muscles flaccid—no glottic reflex—apex beat failing.

II. APGAR RATING[13]

This is an evaluation given the baby 60 seconds after birth and subsequent times thereafter. Values of 0, 1 or 2 are given to five aspects of the child giving a perfect score of 10. See table two.

III. SOME SIGNS OF FETAL DISTRESS

1. Intrauterine.

(a) Bradycardia of the fetal heart rate—any drop of 20 points is significant.

(b) Excessive activity may signify a hypoglycemic state which is corrected by intravenous glucose to the mother.

(c) Meconium staining the amniotic fluid except in a breech presentation is indicative of a low oxygen saturation and therefore fetal anoxia.

2. Extrauterine.

(a) Tachycardia, cyanosis, retraction of the soft parts of the chest, progressive restlessness to convulsion and death is indicative of an obstructed airway.

(b) Constant coughing or undue distension of the abdomen may indicate a tracheal oesophageal fistula.

Table Two

	2	1	0
Heart Rate	100–140	under 100 over 150	none
Respiratory Effort	Cries lustily	irregular and shallow	apnea over 60 secs.
Reflex Irritability	sneezes and coughs on suctioning—grimaces	slight response to suctioning	none
Muscle Tone	resists motion moves about	variation	flaccid
Colour	pink	blue extremities	blue

Ratings for a normal centre	Deaths
0 1 2	14%
3 4 5 6	1.1%
8 9 10	0.13%

Therapeudic Aspects

I. prevention of asphyxsia

1. Antenatal.

Early examination is important so that education of the mother and any corrective measures to her health may be started early. Proper diet and weight control is important. Early recognition of multiple pregnancy, preeclampsia, placenta previa and other conditions requiring special care are important.

2. Conduct of Labor.

First stage of labor.

(a) Assess the patient and decide on the type of delivery.

(b) The baby must be watched for signs of fetal distress. It is important to deliver the infant before anoxia becomes severe.

(c) Induction of labor;

Indications:

1. Induced before term in preeclampsia and active hemorrhage. Special consideration is given to cases where previous erythroblastosis foetalis occurred. Here induction may be performed at a date just before the previous stillbirth in hopes the early delivery may prevent destruction of the baby.[14]

2. Fetal distress.

3. Maternal weight falling in women carrying post mature infants.

4. Primary uterine inertia.

Methods currently used:

1. Surgical rupture of membranes has been found satisfactory without increasing the incidence of resuscitation.

2. Pitocin drip is not a doctor's convenience but a tool. It may be used when other methods fail. It does increase the need of resuscitation.

Pitocin drip is contraindicated in the following:

(a) Any gravida four.
(b) Any dystocia.
(c) Any abnormal presentation.
(d) Breech presentation.
(e) Multiple birth.

(f) Floating head.
(g) Fetal distress.

The doctor in charge of the patient should do these procedures and be available at all times thereafter until the delivery of the baby.[15]

(d) One must choose an analgesic. Many people use nisentil 40 mgm as it is rapidly destroyed. Allyl-normorphine hydrobromide (nalorphin) is a drug which is antagonistic to morphine, methadone, dilaudid, metafor, and pethidine thus reopening the opiate analgesics to the obstetrician. It is, however, ineffective against barbiturates, anaesthetic drugs and physiological causes. Given alone it is a respiratory depressant. In one series it has been effective in reducing the number of babies needing resuscitation by 52%. No ill effects have been noted. Most babies cry or gasp within one half to two minutes of injection. It is heartily endorsed by many authors.[16, 17, 18]

Method: With demerol as analgesic give 10 mgm nalorphin I.M. to the mother ten minutes before suspected delivery. As the baby is born inject 0.10 mgm to 0.25 mgm into the umbilical vein. This may be repeated if necessary.

(e) Each anaesthetic has its own dangers, whether inhalation, spinal or caudal block, or regional block. It is important that, during the anaesthetic the mother's blood pressure is not allowed to fall and that at all times she is well oxygenated.

(f) All maternal medications should be reported on the baby's chart and also any peculiarities which occurred during the delivery.

Second stage of labour.

The following steps should be taken if possible as they tend to reduce fetal anoxia:

(g) Slow delivery.

(h) Prophylactic outlet forceps if necessary with a large episiotomy.

(i) Suction the nares and the glottic areas during delivery.

Third stage of labour.

It is advocated that the obstetrician milk the umbilical cord six to ten times in order to afford maximum blood to the fetus. In so doing he will increase the oxygen carriage, the blood pressure and the baby's iron stores, It is, however, contraindicated in blood incompatability and transplacental narcosis.[19]

II. RESUSCITATION

Speed is a virtue but gentleness is essential. We are dealing with a baby that is in shock, not a football player.

1. Aspiration and clearing of the air passages.

(a) Nasopharyngeal aspiration started during delivery is completed by clearing the mucus out of the glottic area thoroughly—before respiration if possible.

(b) Tracheal and bronchial aspiration necessitates the use of a laryngoscope. It must be noted that although mature infants stand this operation well, prematures are very susceptible to shock and it is therefore wise to delay tracheobronchoscopy in their case.

Method: A size 14 French catheter with extra suction holes under 3–5 mm. Hg suction force is introduced down the trachea under direct vision afforded by the laryngoscope.

(c) Gravity is used to promote drainage and 15–20 degrees Trendelenburg is used. Giant swings and such are to be condemned.

2. Continuous and/or intermittent positive pressure by mask:

Continuous oxygen by nasal insufflation has been found to be an inefficient method of giving oxygen, but a mask

does so effectively and with relative ease. The mask may have a tab to hold the tongue forward; others require an oropharyngeal airway. Often no airway is used, but this is not without danger, and one must watch for obstruction by the tongue.

There are two current theories on the use of intermittent positive pressure.

(a) Day[20] concluded from his work that a certain minimum pressure must be exerted to correct atelectasis and in employing this pressure safely, its duration must be limited. Rupture of the alveoli was a result of overdistension from too high a pressure, or too long an application of any pressure. He proceeded to demonstrate that higher pressures, applied for short periods of time, could be given with expansion of lung tissue, whereas lower pressures, used for a longer duration, did not. (Both without damage to the alveoli.)

Goddard[21] obtained the following results from stillborn lungs:

Cm. of water	*Degree of expansion*
20	no expansion
30	patchy expansion
40	fair expansion
50	uniform expansion
60	marked expansion
75	over distension
95	rupture

He also found that 2–3 times the tidal volume of air could be introduced into the lungs of small prematures without any rupture of alveoli. From this he concluded that the lungs had amazing elasticity. He suggests the following method, using a mask.

1. Initial pressure 40–50 cm. water at 0.2 sec. duration for 12 impulses with an expiratory interval of 0.4–0.8 sec.
2. Suction.
3. Reduce pressure by 20 cm. of water for 12 impulses.

Once the baby starts to breathe switch to continuous oxygen.

Under this regime, less than ten percent needed intubation (in a series of babies all needing resuscitation). This method is not accepted in all centres. Authors using this method, however, have not had any harm occur to the babies which could be attributed to the method.

(b) Wilson[22] concluded from his work with balloons that another factor was present. He contends that the rupture of alveolar tissue and hemorrhage resulting from artificial respiration is dependent not only on the degree of pressure and its duration of application, but also on the proportion of expansile lung to unexpanded lung. He postulates that the damage is caused by the expandible lung filling not only its own space, but also that volume designated for the unexpanded lung.

With this in mind many people advise the use of pressures of 10–20 cm of water given to the baby and then change over to continuous oxygen as the infant starts to breathe on its own.

3. Intubation.[23, 24]

Intubation is used if the baby is flaccid or has an Apgar rating of less than 5. Do not intubate if the glottic reflex is present. Prematures tolerate intubation badly.

Method of intubation:

(a) Place the infant flat on its back with its head on the table.

(b) Insert the tip of the laryngoscope gently to the glottis.

(c) The handle of the laryngoscope is lifted vertically bringing the glottis into view. (Do not lever on the upper jaw.)

(d) Suction and insert the endotracheal tube (Magill 00 or 000). Be gentle.

Intubation is done to insure a clear airway.

4. Electrophrenic stimulation.

This method is still in an experimental

stage. It is agreed, however, by most authors, that it shows promise and may have a place in resuscitation, but as yet it is not practical.[25]

5. Gastro intestinal oxygen.

This method is used to temporarily supply oxygen to the child and so alleviate anoxia which occurred during delivery. It is not traumatic and it is believed to be very useful in the resuscitation of premature infants. It has also been used to combat cyanotic attacks in the post delivery phase. It is highly recommended by many authors.[26, 27]

METHOD

(a) The stomach is emptied by a 14 French catheter which is left in place as an outlet tube to oxygen and so prevent distension.

(b) A second tube, usually a one mm. polythene tube is introduced into the stomach and oxygen at 1 L. per minute is delivered.

(c) Other resuscitative measures may occur concurrently.

In a few moments the child's cyanotic colour will show improvement.

6. Hypothermia.

Experiments show that hypothermia increases the tolerance to anoxia in animals. As yet no human tests have been reported. The majority of authors recommend a constant, normal body temperature in infants to reduce the shock of delivery.[28]

7. Analeptic drugs.

These have no use in resuscitation and are condemned by most authors.

8. Carbon dioxide.

Carbon dioxide is of no value in resuscitation but may be of value in the nursery.

In practice none of the machines have yet proven to be of real value and it is generally accepted that the most important aspects of resuscitation are a clear airway, warmth, and oxygen.

III. POST DELIVERY AND NURSERY STAGES

1. Incubators with special features other than continuous oxygen and warmth.

(a) Bloxson Air Lock.

This is a box incubator in which pressure changes may be cycled. Apgar and Kreisilman[29] in their series could find no advantage in using this over simple incubators.

(b) Eve Rocking Bed Incubator.[30]

This machine tilts the incubator, thus allowing a pumping action of the abdominal contents to aid in respiration. Authors are not all in accord with this incubator. Those who do advocate it, state that prematures, in whom respiration is minimal, benefit. Whether this is due to a constant stimulus to the baby or an actual increase in excursion of respiration is not known. It is useless if the airway is not patent and is of no advantage to the child who makes vigorous efforts on its own.

(c) Drinker.[30]

This incubator is useful only if the child is breathing. It can be useful in increasing the excursion of the thoracic wall.

2. Before the baby leaves the delivery room:

(a) Evaluate the child.

(b) Note the cry.

(c) Note the respirations, checking for rib retraction.

(d) Note any gross abnormalities and report these.

(e) Check the tongue and be sure it is not blocking the airway.

(f) At the nursery give the baby to someone responsible. Arrange for competent nursery supervision.

3. At the nursery.

Minimal handling is essential in good nursery care. If there is no damage to vital centres, there is a tendency to rapid and spontaneous recovery if left alone.

Routine care.

(a) Maintain postural drainage (10 to 15 degrees Trendelenburg) frequently changing the baby's position from side to side to promote drainage.

(b) Avoid regurgitation of gastric contents. For the first twelve to twenty-four hours give nothing by mouth. If the baby is dehydrated give a clysis. If the baby is drooling excessively, aspirate the stomach contents. Little,[31] in a series of 400 Caesarean sections (200 controls), using gastric aspiration, decreased the mortality by 4.4% in the aspirated group.

(c) Oxygen is advocated by most authors for babies having difficulty. It is important that the oxygen does not attain values over 40% in order to prevent retrolental fibroplasia. The delivery of oxygen is best if bubbled through warm water so as not to disturb the temperature in the incubator. When weaning a baby from oxygen do so gradually and not all at once.

4. Special considerations

(a) Cyanotic attacks may occur in the nursery in this period. If they do:

1. First check the airway and start oxygen into the incubator.
2. As mentioned before, gastric oxygen may be used as long as 12–24 hours.

(b) If an increase in respiration is desired some authors advocate the use of carbon dioxide using the following as a guide:

1. Too much carbon dioxide causes the baby to have grunting respiration.
2. Too little carbon dioxide and the baby's respiration will be quiet or apnoeic.
3. A starting point commonly used is 5% carbon dioxide in oxygen or air delivered to the incubator.
4. Make sure that the carbon dioxide does not build up to high concentrations.

(c) Love and Tilley[32] observed that the indrawing of the chest cage with contraction of the diaphragm in small newborns was similar to the paroxysmal respiration found in adults suffering from crush injuries of the chest. They recommend suspension of the chest wall by a towel clip inserted in the fascia of the sternum attached to elastic bands to the top of the walls of the incubator. This stabilizes the thoracic cage and allows for more efficient diaphragmatic respiration. They have used this method as long as 72 hours until less negative intrathoracic pressure is needed to overcome the surface tension of moist alveoli. Many authors recommended this method as worthy of trial.[33]

(d) Most authors recommended a moist atmosphere as a preventative measure against hyaline membrane disease and to liquify bronchial secretions. Detergents and enzyme products have been devised to increase the efficiency of this mechanism. Despite early encouraging reports these materials have not changed the prognosis of hyaline membrane disease and in controlled series[34] had no more benefit than a high humidity.

How long should one treat asphyxia neonatorum when it occurs? The fetus at birth can survive possibly 30 minutes without oxygen as long as its circulation remains intact, but it is doubtful whether it survives unscathed. Donald estimated that after 15 minutes of anoxia irreversible changes have occurred in the central nervous system.

In a series[35] of 500 children admitted to hospital for cerebral symptoms including cerebral palsy, mental deficiency, and epilepsy, 70% had a history of asphyxia neonatorum. These results are supported by other authors[36] in this field.

Anoxia is followed by vasomotor and respiratory depression which can in turn cause further anoxia, and ir-

reversible changes soon set in as a vicious cycle is established. It is to be hoped that with better understanding of this problem a more satisfying solution may be found to treat asphyxsia neonatorum.

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SOME PHYSIOLOGICAL AND CLINICAL ASPECTS OF SHOCK

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THE problem of shock remains a challenge for investigators today. With the increased pace of our modern mechanized age and its inevitable accidents, shock is growing in peacetime importance. In the following discussion two aspects of the problem will be reviewed; the physiological mechanisms thought to be involved in shock, and the clinical appearance of the patient in shock. It is necessary to restrict this review to a discussion of shock, secondary to injury.

The term shock, secondary to injury, means that condition of progressive circulatory deterioration, following in a relatively few hours after some hemorrhage or trauma and if untreated, leading to a fatal termination. It is known that the condition may reach a so-called irreversible state, after which fluid therapy is of no value.

Physiological Mechanisms

The physiological changes occurring when the process of shock is initiated in a patient are intriguing to the research worker. They are of great importance, also, to the clinician in his interpretation of his physical findings.

I. vascular phenomena

Emphasis will be placed on the vascular changes as a knowledge of them may be most advantageously applied to the interpretation of the findings in the patient.

One of the few points concerning shock about which physiologists agree is that the initiating factor of the process is, for all practical purposes, a decrease in the effective circulating blood volume. The decrease in volume may be a result of the loss of whole blood, plasma or fluid from traumatized tissue in burned or crushed areas. Hemorrhage, of course, is the commonest cause, particularly in wartime. Loss of plasma or fluid may be less obvious. It is lost through seepage in the injured area. There is no proof of a generalized decrease in capillary permeability as an early event leading to loss of fluid from vascular beds in areas other than those injured.

Studies made by Evans[1] during the Second World War on a number of wounded men in shock from trauma and hemorrhage, using a dye-hematocrit method for blood volume determination, showed that a decrease of fifteen percent of total blood volume was sufficient to cause development of shock. Beecher[1] in 1949, using similar methods, found that a reduction of one third of total blood volume was necessary to produce moderate shock, a reduction of one-half leading to severe shock. It is thought that after tissue trauma, less reduction in blood volume is required to produce shock than in shock caused by hemorrhage[1] alone.

Although it is not the purpose of this article to deal with such cases, it should be noted that conditions leading to excessive diarrhoea, vomiting, dehydration due to diabetes, electrolyte shifts such as those found in adrenal cortical insufficiency and many others, can result in a somewhat similar syndrome due to an initial decrease in effective circulating blood volume. The physical examination of these patients will be complicated by the manifestations of the disease that has led to the development of shock.

As a result of the decrease in blood volume a cycle of circulatory events is initiated, which, if uninterrupted by therapeutic measures, will proceed to an

irreversible stage and death of the patient. With less eireulating blood there is a deerease in the pressure gradient between the peripheral and central veins and consequently less blood is returned to the right side of the heart. With less blood returning to the heart, the length of the eardiae musele fibres is shorter at the onset of systole. There is, also, less blood available for ejection from the heart on systole. The decrease in the initial length of the cardiac fibres lessens the force of contraction of each heart beat (Starling's Law). Since the stroke volume is diminished, a marked decrease in cardiac output results. This drop in cardiac output would be reflected in a diminution in arterial pressure, were it not for eompensatory vasoconstriction.

Superimposed upon the circulatory changes already mentioned are a variety of compensatory mechanisms. A fall in central arterial blood pressure acts as a stimulus to the sensitive carotid sinus and aortic areh. From these receptors, reflexes are initiated, whieh pass through the cardiac centers in the medulla and the vasomotor eenters, resulting in an increased heart rate and in constriction of the peripheral vascular beds and dilatation of those in the coronary and eerebral areas. This vasoconstriction of sympathetic nervous system origin is accompanied by a generalized stimulation of sympathetie fibres.

The result of the compensatory inerease in heart rate and vasoeonstriction is, in many patients, at least a temporary maintenance of peripheral arterial blood pressure. It must be remembered that blood pressure, under these circumstanees, is being maintained at the expense of the blood supply to eertain organs and tissues. As the eireulation deteriorates the benefieial nature of this vasoeonstrietion becomes debatable.

In the absence of adequate therapy, circulation to the brain eventually deteriorates in spite of the efforts of the compensatory mechanisms. Once the cerebral centres become anoxic, the compensatory mechanisms fail and the patient is liable to show, in late stages of shock, collapse of peripheral resistance and slowing of the heart.

II. RESPIRATORY PHENOMENA

The respiration of the patient in shock is largely dependent on the state of his circulation. The fall in blood pressure in the carotid sinus and aortic arch acting on the respiratory centers leads to a reflex increase in respiratory rate. The decrease in oxygen content of arterial blood will act on the respiratory centre directly, to a eertain extent, and on the chemoreceptors of the carotid and aortic bodies to cause an increase in respiratory rate and depth.

The total volume of oxygen carried to the tissues, dependent on the cardiac output and the oxygen content of arterial blood, is usually reduced by more than half. However, owing to the stagnant anoxia assoeiated with shock, the percentage of oxygen extracted by the tissues is greatly increased. The liver, since it depends largely on desaturated blood for its oxygen supply, will suffer from lack of oxygen early in the process.

III. METABOLIC PHENOMENA

It is difficult to tell if the metabolic changes result from or contribute to the circulatory and respiratory changes. It has been established that, by mechanisms varying in different types of shock, the circulating blood volume is decreased and with this decrease, there is a diminution of blood flow. This diminution results in a diminished supply of oxygen to the tissues. It has been suggested[10] that the first tissues to be affeeted in this way are probably those of the extremities and the liver. In

general the metabolic phenomena observed in shock are those one would expect in an anaerobic type of metabolism. Factors other than anoxia, including an increase in the adrenal hormone level of the blood, will influence metabolic changes. The metabolic changes that have been measured have been studied in experimental animals and have been related mainly to the function of muscle and liver. Glycogen storage by the liver is impaired and the blood sugar level will rise, in shock, if glycogen is present in sufficient quantities initially. The level will fall as soon as these stores are depleted. It is also thought that glucose utilization may rise.[10] This fact is supported by the knowledge that the glycogen of muscle is depleted. A rise in lactate and pyruvate of the blood as shock progresses, also points to an anaerobic type of metabolism.

Further evidence favouring distortion of the functions of the liver has been found in the failure of that organ to deal adequately with the increased protein catabolism of the anoxic and traumatized peripheral tissue.

The significance of these and many other metabolic changes in shock has not yet been clarified. In general they suggest at least an alteration in liver function and a shift toward an anaerobic type of metabolism. A point of interest here is that the efficiency of production of energy in anaerobic metabolism is approximately one twelfth of that in normal aerobic metabolism.[7]

Physical Appearance of the Patient

It must be remembered on examination of the patient suspected of being in shock that there are no specific pathognomonic signs. The signs and symptoms related to the passage of a ureteral or biliary stone may show a remarkable similarity to those seen in a shocked patient, revealing a feeble, rapid pulse, pallor, low blood pressure, rapid breathing and extreme muscle weakness. In describing shock, Watson-Jones[3] writes ". . . it is a complex of syndromes in which protective reactions and lethal consequences are distinguished only with difficulty."

I. general appearance

The most striking feature on first observation of the patient in well advanced shock is usually a pallor of the skin, described as ashen grey in many texts. The decrease in colour intensity is explained by peripheral vasoconstriction; the blue-grey colour, if present, is explained by the stagnant anoxia which leads to an elevation of the reduced hemoglobin in the blood.

As a result of the generalized sympathetic nervous system response in shock, the patient frequently sweats profusely, regardless of the environmental temperature.

The patient seldom makes purposeful movements since extreme fatigue follows depression of metabolism. He is frequently restless, however. General discomfort and a decrease in blood supply to the central nervous system probably help to produce this restlessness. It has also been observed that the patient is seldom in extreme pain.

Except in the late stages, when anoxia of the respiratory centre leads to depressed breathing, the patient is usually breathing deeply and rapidly. This type of respiration would be expected as a result of the decrease in blood pressure and oxygen level of the blood.

Occasionally, vomiting and diarrhoea are observed. Any fluid lost in this way would contribute to the decrease in the effective circulating blood volume.

II. blood vascular observations

(a) *Blood Loss.* The initiating factor of the shock process is a decrease in the effective circulating blood volume. In most cases of external hemorrhage, this

loss will be obvious. However, detection of plasma or fluid loss may be difficult. An increase in the diameter of the thigh of two centimetres can indicate the presence of approximately one litre of fluid. Trauma to the abdomen caused by blunt objects may result in the loss of a large amount of blood into the peritoneal cavity and, when injury to the thorax or head is observed, distortion of the usual shock syndrome may be anticipated.

With the loss of fluid, dehydration of the tissues often results. When the buccal mucous membranes become dry, the patient complains of thirst, the severity of which intensifies as the syndrome progresses.

(b) *Veins.* The peripheral veins frequently show an abnormal degree of collapse due to peripheral vasoconstriction and the decrease in effective circulating blood volume.

(c) *Pulse.* The pulse of the patient in shock is aptly described as rapid and thready. Since it reflects heart rate, the pulse will be rapid if the compensatory rise in heart rate materializes. The drop in cardiac output will decrease the volume of blood reaching the peripheral vessels with each heart beat causing a weak, often barely palpable pulse. If the individual possesses a sensitive vagus nerve system, his heart rate and pulse may be normal or slow throughout the course of the syndrome. In the late stages, as the cerebral centres become anoxic, the pulse will become slower. This is a grave prognostic sign.

(d) *Blood Pressure.* In discussing the physiology of the compensatory increase in heart rate and peripheral resistance found in shock, it was pointed out that the blood pressure may be maintained at a normal or, occasionally, at an even greater than normal level. Since visceral and peripheral circulations are sacrificed to maintain the pressure, the compensatory mechanism, if overactive, may hasten metabolic deterioration and irreversibility. Low blood pressure is more commonly associated with shock and, if prolonged, it is a serious sign. When transfusions produce only temporary elevations of pressure an irreversible state of shock should be suspected. Wiggers[1] feels that a fall in pulse pressure is a more significant finding than an overall decrease in blood pressure. Since diastolic pressure is maintained largely by arterial elasticity, it will not be affected to the same extent by the diminished cardiac output as will the systolic pressure. It is clear that, although a single blood pressure reading may bear no significance, a constant alertness for changes in blood pressure is necessary in the care of the patient suspected of being in shock. The conception of shock as a progression of effects is particularly applicable to the blood pressure. However, if one is to intelligently understand the problem of shock, this concept must be applied to all phases of the syndrome.

(e) *Renal Blood Flow.* With constriction of the peripheral vascular beds, blood flow to the kidney decreases. The patient in shock shows oliguria and frequently anuria. The fact that the urine, if some does flow, is not concentrated, is significant. This finding suggests that the decrease in urine volume should be attributed to the decrease in renal blood flow.

III. BODY TEMPERATURE

In shock, blood flow to the skin is decreased and when the skin is touched it is usually described as cold and clammy. The skin temperature is usually decreased and vasoconstriction of the oral mucous membranes results in a lowered oral temperature. Consequently, in the past, artificial warming of the patient was advocated. The cutaneous vasoconstriction has been recognized as a compensatory mechanism. Wright and Devine[8]

have found that, provided that the environmental temperature is not excessively low, rectal temperatures of untreated patients in shock are usually normal and frequently above normal. This fact is reflected in the absence of shivering in the patient in shock, since shivering is initiated by a lowering of the temperature of the blood in the vessels supplying the temperature regulating centres in the hypothalamus.

IV. INDIVIDUAL VARIATION

It is hoped that the possibility of the wide variations in the manifestations of the shock syndrome has been made clear. The varying degrees of compensation can produce a wide range of clinical signs in the patient. It is interesting to think that certain types of humans may be more susceptible to shock than others. No doubt, experienced clinicians have their own opinions on this subject on which they rely in judging the patient in shock.

Generally speaking, it has been found that changes associated with the aging process increase the tendency toward collapse and shock. Poor nutrition, pain, infection, hunger, dehydration and fear also predispose to shock.

CONCLUSION

The most important consideration in the examination of the patient suspected to be in shock is the early recognition of the condition. It is essential that the condition be recognized before the onset of irreversible changes in order that therapeutic measures may be taken. A history of some trauma or blood loss is of course an invaluable aid since, at the onset of shock, none of the classical physical signs may be present. Experience is, therefore, a vital factor in the examination of the patient in shock. However, if experience is to be accumulated intelligently by the novice, it is important that he should be able to interpret what he sees, in terms of the physiological mechanisms at work in the patient.

ACKNOWLEDGMENT

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THE PROBLEM OF SUBDURAL HEMATOMA

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IN the field of neurology subdural hematoma is now recognized as "the great imitator".

During July, 1955, there were admitted to the public neurosurgical service of the Toronto General Hospital eight patients in which the diagnosis was ultimately proven by operation to be subdural hematoma. Six of these were on the ward at the same time, and study of these six illustrates the need for constant awareness of the possibility of a subdural hematoma: important points in diagnosis and management are considered.

Case Histories

I. W. R., a forty-six year old clerk, was found in a deep stupor on his living room floor. He was known to drink but there was no indication that he had been drinking at this time, nor was there any evidence suggesting a blow on the head. On admission to another hospital he responded only to painful stimuli. Blood pressure was 180/110, pulse was 80, and temperature was 104°F. Neurological examination revealed normal pupils with no papilledema, absent reflexes in both legs, a depressed level of consciousness. During the subsequent four days he had four major convulsions, but between attacks his threshold to stimuli remained as before.

The patient was then transferred to this service where such causes of unconsciousness as renal failure, overdosage of drugs, cerebrovascular accident, and meningitis were ruled out as completely as possible by carrying out a careful physical examination, urinalysis, and routine blood work. Since there had been no improvement in the patient's condition in the first four days and since a subdural hematoma was a definite possibility, bilateral burr-holes were made, revealing a large right-sided subdural hematoma, which was evacuated.

The patient continued in coma for three days after the operation and then gradually recovered full consciousness over the next week. He was discharged with slight weakness of the left leg as the only neurological defect remaining. At this time careful questioning still did not elicit any history of trauma to which the illness could be attributed.

This was a case of unconsciousness of unknown etiology in which subdural hematoma became a likely diagnosis by a process of elimination.

II. W. S., a vigorous seventy-one year old farmer, had severe frontal headaches for three months, transient dizzy spells for four days, with confusion and disorientation. He was taken to the local hospital and on examination considered psychotic.

He was transferred here and examination revealed weakness of the dorsi-flexors of both ankles and moderate mental confusion. During the next eighteen hours he became drowsy and irritable, and then deeply stuporous. His respiration became Cheynes-Stokes in character, and his pulse fell to forty per minute. There was neither rise in blood pressure nor papilledema, and his pupils remained normal.

Skull X-rays did not demonstrate a calcified pineal body, but an electroencephalogram (E.E.G.) revealed an abnormal focus over the right frontal lobe. The tentative diagnosis of subdural hematoma was confirmed at operation when a large clot with a thick-walled membrane was removed from the right frontal region.

Postoperatively, the patient was quite conscious but confused and euphoric for two days. Gradually, over the next few days, he became normal except for amnesia for the two weeks preceding the operation.

At no time was he or his wife able to recall his receiving a blow on the head.

In this case a psychotic personality trend was shown finally to be due to a subdural hematoma.

III. H. W., a fifty-four year old labourer, was admitted because of increasingly severe right frontal headaches and dizzy spells, and also nausea and vomiting which were worse on standing up suddenly or looking to one side. Severe motor ataxia had been present for one week. No history of trauma could be elicited on careful questioning. Slurred speech, bilateral

up-going toes, and a motor ataxia were the only positive findings. There was no papilledema.

Skull plates again did not reveal a calcified pineal gland but an E.E.G. showed generalized suppression of activity on the right side. An air-encephalogram was also done demonstrating a shift of the ventricles to the left.

At operation a chronic subdural hematoma was evacuated from the right side. In two weeks the patient recovered completely except for slight weakness of the left leg.

In this case frontal lobe tumour and cerebellar disease were considered the most likely diagnoses but subdural hematoma could not be ruled out. Burr-holes were the only way of establishing the diagnosis.

IV. M. W., a seventy-three year old housewife, had been hit by a car and was in coma when admitted. During the next twelve hours, consciousness improved but deteriorated again during the next two days. Apart from her depressed level of consciousness, an up-going right toe was the only significant finding. There was no papilledema and the pupils were normal.

At operation a subdural hematoma without an obvious membrane was found on the left side and was evacuated.

The patient made an uneventful recovery during the next two weeks and on discharge suffered only from amnesia for a period of a few days before the accident.

This was a case of an acute subdural hematoma. This lesion cannot be differentiated preoperatively from an acute extradural hematoma, a rarer disease.

V. L. L., a thirty-four year old machinist, fell and struck his head on a cement wall and was knocked out for a short time. For the next three months he had continuous, throbbing, frontal and bi-temporal headaches, worse in the morning and evening and preventing sleep. Coughing aggravated the headache. Anorexia and a resulting loss in weight of fifteen pounds occurred and there was severe vomiting for the two weeks prior to admission. There were no helpful physical findings but, during the next four days while in hospital, he became less alert, his blood pressure rose from 115/75 to 150/90 and questionable papilledema in the left eye was noted. The hypertension was not associated with a bradycardia and at no time were there pupillary changes.

X-rays revealed the pineal body to be shifted one cm. to the right of the mid-line.

Burr-holes revealed a chronic subdural hematoma and rapid and complete recovery followed its removal.

Of the examples given in this paper this most closely resembles the typical text-book history of a chronic subdural hematoma, and it is important to realize that operation, if done early, before there is any major neurological disorder, will almost invariably result in a complete and surprisingly early recovery.

VI. F. L., a seventy-two year old spinster, fell on her verandah steps, striking her right forehead. Consciousness was not disturbed. She remained at home in bed for the next twelve days, feeling very tired, vomiting several times and having occasional right frontal headaches. After admission she became progressively more drowsy and three days later it was noted that her left plantar response had become extensor, and her systolic blood pressure had risen from 140 to 200. This was unaccompanied by a significant change in pulse rate, or in pupils, and there was no papilledema.

Skull X-rays revealed a centrally situated pineal gland. E.E.G. did not help.

When bilateral burr-holes were made, a large right-sided subdural hematoma and a smaller left-sided one were discovered and evacuated.

In eight hours the patient had responded well and was well orientated, thereafter making an uneventful recovery.

This illness, with a course of two weeks, is a typical case of subacute subdural hematoma.

Comment

These six cases help to show that, if a subdural hematoma cannot be excluded, diagnostic bilateral burr-holes must be done.

In Case I no diagnosis other than a subdural hematoma could be established to account for coma and seizures when all the evidence had been accumulated. In Case II an inexplicable psychosis going on to coma led to the diagnosis. In Case III the diagnosis lay between a

frontal lobe or a cerebellar tumour, or a subdural hematoma. In none of these three cases was a history of trauma available. Cases IV, V and VI were fairly representative examples of acute, subacute, and chronic subdural hematomata, each with a clear-cut history of trauma. Case VI exemplifies the fact that in some ten per cent of cases chronic subdural hematoma are bilateral.

A classic symptomatology of increased intracranial pressure was observed in one of our six cases, although some of the elements of it were present in each—e.g. headache, nausea, vomiting, lowered consciousness, rise in blood pressure, slowing pulse rate, papilledema. In only two cases was there any clinical indication to help in lateralizing the lesion, and in both of these an up-going toe suggested the contralateral side as the site of the clot (as shown later this cannot be taken as a definite indication of the side of the clot). In one of these two, the history of trauma and the location of the headache also suggested the side involved. Of the other four, X-ray identified the side of the clot in one, while the E.E.G. did so in two others. In one the clot was not lateralized until operation.

Alcoholism plays a major role in the histories of many subdural clots, as it does in one of ours. It is not thought to have a direct effect but rather exerts this influence by way of the more frequent blows on the head happening to these people.

Diagnosis

I. HISTORY. Although trauma is the accepted cause of subdural hematoma, only in about one quarter of cases can a history of trauma be elicited. This percentage is higher if relatives are consulted also, as they frequently remember minor accidents which the patient himself has forgotten. Hence failure to recall a blow on the head should not lead the investigator astray, as the injury which did the damage may have been very trivial.

In a carefully taken history there are almost certain to be symptoms belonging to one of the following groups:

1. *Increased intracranial pressure* causing headache, vomiting, bradycardia, hypertension, and other such signs and symptoms.
2. *Local disturbance of brain function* resulting in weakness of an arm or leg, sensory loss, or even Jacksonian fits.
3. *General disturbance at a higher level* causing a personality change, irritability, and eventually a lowered level of consciousness leading to coma and death.

II. EXAMINATION OF THE NERVOUS SYSTEM.

1. *Mental State.* Any of the manifestations of general disturbance of cerebral function as described above may be noted on examination. The most important of these is a lowered level of consciousness.

2. *Cranial Nerves.* The only cranial nerve affected at all frequently is the oculomotor nerve. An enlarging, less reactive pupil may occur on the same side as the clot because of herniation of the hippocampal uncus between the tentorium cerebelli and the brain stem, damaging the third nerve. Diplopia can also occur. Defects of the visual fields are rare.

3. *Motor System.* Hemiparesis may occur. This is usually on the side opposite to the clot and is caused by pressure of the clot on the motor cortex. However, it may be on the same side if there is enough deviation of the brain to cause severe pressure by the edge of the tentorium on the contralateral basis pedunculi (Kernaghan's notching). Hyperre-

flexia, clonus, and a Babinsky response may all go along with the weakness.

4. *Sensory System.* Sensory changes may occur, but are much less common than motor changes alone. These are due to pressure on the sensory cortex. The general signs of increased intracranial pressure are usually late, and, in most cases, the diagnosis should be made long before these occur.

III. INVESTIGATION.

1. *X-ray.* A shift of a calcified pineal body is evidence of a one-sided space-occupying lesion. However, bilateral hematomata may result in a centrally placed pineal or in a shifted pineal, depending on the relative sizes of the clots.

2. *Electroencephalography.* An E.E.G. is of great help in some cases where it lateralizes the lesion.

3. *Air Encephalography.* An A.E.G. is of help in doubtful cases, when it may demonstrate a shift of the cranial contents, and also can frequently help differentiate the lesion from a tumour. This technique is, of course, only practicable on a neurosurgical service.

4. *Burr-Holes.* This is the most important diagnostic, as well as therapeutic, procedure. Few realize that burr-holes are a diagnostic method and form the best means of excluding this lesion in the confused or comatose patient. In a neurosurgical operating theatre this is not regarded as a major operation.

5. *Lumbar Puncture.* This is mentioned only to be condemned. Even if there is no papilledema, no hypertension, and no other evidence of increased intracranial pressure, such a procedure is still very dangerous, and possibly fatal, in a case of subdural hematoma. Sudden decompression from below may allow herniation of a tight brain through the foramen magnum, causing death from brain stem disruption.

History, physical examination, X-ray and E.E.G. will diagnose the condition in most cases. If there is still any possibility of a subdural hematoma bilateral burr-holes should be made.

TREATMENT

Careful nursing, adequate pre-operative and postoperative hydration and nutrition, bowel and bladder care, the conservative use of sedation, and the traditional McKenzie routine, are all of the greatest importance in these cases.

Strong emphasis must be placed on this fact: there is only one way to treat a subdural hematoma—complete evacuation. Bilateral burr-holes, followed by evacuation of the clot and incision of the deep membrane in a cruciate fashion is the procedure of choice. If the bilateral approach is used routinely no extra post-operative morbidity occurs and the small subdural hematoma on the unsuspected side which later gives trouble, is rarely missed.

(Diagnosis and management of subdural hematomata in infants have not been considered in this paper.)

PROGNOSIS

Most cases are cured completely.

Only when diagnosis is late and there is severe neurological deficit before operation is some residual abnormality to be expected.

Of our six cases none died. Two of the six were left with slight weakness of one leg which may recover in time. The remaining four patients are now completely well.

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We wish to express our appreciation to Dr. E. H. Botterell for his encouragement and help in writing this paper.

ABSTRACTS

A Comparison of Cortisone and Aspirin in the Treatment of Early Cases of Rheumatoid Arthritis: The Joint Committee of the Medical Research Council and Nuffield Foundation. B.M.J. **4941:** 695, 1955.

This is the second report submitted at the end of the second year of trials on 61 adult patients chosen for treatment at six centres in England and Scotland; 30 were on cortisone and 31 on aspirin. The criteria used to select the patients were as follows:

1. Polyarthritis of rheumatoid type affecting at least four joints and bilateral involvement of either hands or feet, ankles or wrists.

2. Had the disease for not less than three and not more than nine months.

3. Age between 17 and 59 years.

The two groups of patients were initially almost identical in the numbers of men and women, in the number aged 17–39 and 40–59 years, and in the numbers with a duration of symptoms of 3–5 or 6–9 months.

During the first year, treatment was given in twelve week courses separated by one week off treatment. Each courses started with a standard dose after which the physician employed the minimum individual dose (after three weeks). Since some showed relapses during the rest periods and since this was undesirable, the therapy during the second year was one of continuous treatment at maintenance dosage unless the patient was in remission.

The patient's progress was assessed in two ways:

1. *Subjectively:*

(a) Judgment of the patient's general funetional capaeity.

(b) Activity of the disease as inactive, slightly active or very active.

(c) Patient in remission or not.

2. *Objectively:*

(a) Joint tenderness.

(b) Range of movement and strength of grip.

(c) Tests of dexterity.

(d) Hemoglobin level and blood sedimentation rate.

The first report concluded that "For practical purposes, therefore, there appears to have been surprisingly little to choose between cortisone and aspirin in the management of these 61 patients in the early stages of rheumatoid arthritis." During the second year, only 58 were treated, 30 on cortisone and 28 on aspirin. At the end of two years there was still a remarkable similarity between the two groups in the results obtained both with the subjective and quantitative measurements. Four in each group were in remission, six on cortisone and five on aspirin were "very active," and nine on cortisone and seven on aspirin were still badly incapacitated.

T. Izukawa.

Complications and post-operative care after tracheotomy. Putney, F. J. AMA Arch. Otolaryn. **62:** 272, 1955.

The author's purpose is to give an objective analysis of the complications of this eommon procedure. A summary of the complications arising from the technique and improper after-care is made. He presents two cases to illustrate complieations from both.

Faulty technique is held to be responsible for such complications as mediastinal emphysema and pneumo-

thorax which may be prevented by the preoperative introduction of bronchoscope or intratracheal airway. Pneumothorax is a common complication but in most cases the diagnosis cannot be made without a chest X-ray. Stenosis with delay in decannulation occurs most commonly in infants and young children who have a soft tracheal wall. Causes are tight suturing of the tracheotomy wound leading to pretracheal infection, removal of a segment of a tracheal ring rather than vertical incision of two rings, and tracheotomy above the level of the second ring.

Rarer complications are severe hemorrhage and atelectasis and pneumonitis. The latter can be prevented by maintaining a clear air way and by the use of antibiotics.

For these reasons, the after-care of tracheotomy should consist of regular aspiration of secretions with a soft rubber catheter passed down both bronchi if possible. Encrusted secretions should be removed by the introduction of isotonic saline solution into the trachea before aspiration. Humidification and nebulization of inspired moisture minimize this complication. Delay in development of the larynx in tracheotomized children is prevented by forced breathing through the larynx. The tracheotomy tube should be changed daily to help prevent delay in decannulation.

The author concludes that despite these complications tracheotomy is a justified procedure since most of these complications are preventable.

M. S.

Virus Reproduction and Virus Disease: Horsfall, Frank L., Jr. C.M.A.J. **73:** 778, 1955.

Reed's and Carroll's observations (1902) on yellow fever etiology, and accumulated information on similar virus diseases have allowed the formation of a general scheme for virus disease pathogenesis.

Theoretically a single virus particle in a susceptible cell can initiate virus reproduction, subsequently proceeding by logarithmic increase. The more cells infected and the faster the reproductive rate, the smaller will be the incubation period and the more severe and extensive will be the lesions.

Evidence suggests that recovery is due to a decrease in the number of cells susceptible to virus reproduction, rather than to the appearances of antibodies. Persistent immunity, however, is produced by the action of protective antibodies formed as a result of the persistence of a few slowly reproducing viruses. Virus reproduction without disease is possible, by variance of virus virulence, concentration in cells or reproductive rate—the factors concerned in cellular damage.

Unlike bacterial viruses (bacteriophages) whose reproductive mechanisms have been carefully traced and always result in bacterial destruction, animal virus reproduction is not clear and does not always result in cell destruction. However, bacteriophage reproduction is inhibited by chloramphenicol and tryptophane analogues, which inhibit protein synthesis within bacteria, and there is evidence that animal virus reproduction is inhibited by similar substances, such as methoxinine and other amino acid analogues.

This suggests that inhibition of virus reproduction in human beings is possible, with development of more potent, less toxic drugs. To be effective, these will have to be administered during the incubation period of the disease.

S. Rabinovich

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Easy-to-take, well-tolerated, relatively non-toxic, safe, effective and free from staining associated with gentian violet. Its lack of toxicity when taken in the correct dosage makes it a desirable therapeutic agent.

FOR INFANTS AND CHILDREN

"VERMISOL" SYRUP
Syrup No. 634 "Frosst"

Each 5 cc. teaspoonful contains:
Piperazine hexahydrate..... 500 mg.
(as tartrate)

FOR ADULTS AND OLDER CHILDREN

"VERMISOL" TABLETS
Tablet No. 807 "Frosst"

Each tablet contains:
Piperazine hexahydrate..... 250 mg.
(as tartrate)

DOSAGE:

Infants up to 15 lbs.	½ teaspoonful (2.5 cc.) or 1 tablet	Once daily
Children up to 30 lbs.	½ teaspoonful (2.5 cc.) or 1 tablet	Twice daily
Children 30 to 60 lbs.	1 teaspoonful (5 cc.) or 2 tablets	Twice daily
Children over 60 lbs. and adults	2 teaspoonfuls (10 cc.) or 4 tablets	Twice daily

For Pinworms: Vermisol may be administered for 14 consecutive days or, alternatively, for two 7-day courses one week apart.

For Roundworms: One, 5 to 7-day course of treatment is usually sufficient.

CAUTION: With overdosoge the following reactions may occur: nauseo, giddiness, lock of co-ordination, flashes on closing the eyes, inability to focus vision and o sense of detochment. These untoward effects ore temporary ond disoppear when medicotion is discontinued.

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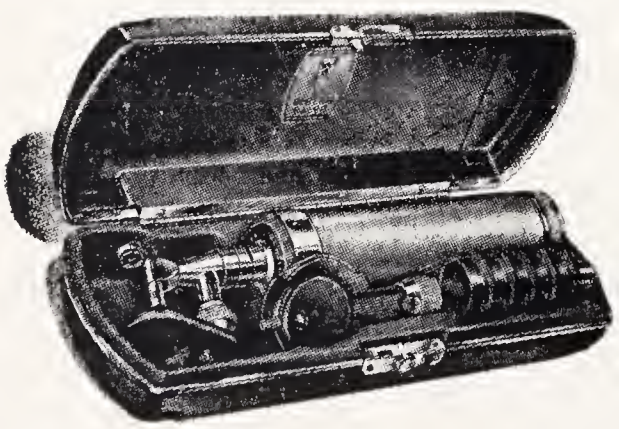

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